How to prevent and how to treat dyspnea in critically ill patients undergoing invasive mechanical ventilation

Gabriel Kemoun[a,b], *Alexandre Demoule*[a,b] *and Maxens Decavèle*[a,b]

Purpose of review
To summarize current data regarding the prevalence, risk factors, consequences, assessment and treatment of dyspnea in critically ill patients receiving invasive mechanical ventilation.

Recent findings
In intubated patients, dyspnea is frequent, perceived as intense, and associated with unfavorable outcomes such as immediate and unbearable distress (e.g. fear of dying), prolonged weaning, and delayed severe psychological consequences (i.e. posttraumatic stress disorders). In noncommunicative patients, dyspnea is named respiratory-related brain suffering (RRBS) and can be detected using dyspnea observations scales. Before initiating pharmacological treatments, nonpharmacological interventions may be tried as they are efficient to alleviate dyspnea.

Summary
As opposed to pain, dyspnea has often been overlooked in terms of detection and management, resulting in its significant underestimation in daily practice. When it is diagnosed, dyspnea can be relieved through straightforward interventions, such as adjusting ventilator settings. Assessing dyspnea in patients undergoing invasive mechanically ventilated may be challenging, especially in noncommunicative patients (RRBS). Implementing a systematic dyspnea assessment in routine, akin to pain, could serve as a first step to reduce RRBS and prevent potential severe psychological consequences. In addition to pharmacological treatments like opioids, a promising approach is to modulate both the sensory (air on the face, trigeminal nerve stimulation) and the affective (relaxing music, hypnosis, directed empathy) components of dyspnea.

Keywords
dyspnea, intensive care unit, mechanical ventilation, posttraumatic stress disorders, ventilator settings

INTRODUCTION

Dyspnea, or the unpleasant and frightening awareness of breathing, ranks among the most common and distressing symptoms that patients undergoing invasive mechanically ventilated (IMV) can experience during an intensive care unit (ICU) stay. Although growing attention has been given to the assessment and treatment of pain in the ICU, very little attention has been given to dyspnea, which remains markedly under-evaluated and under-treated in IMV patients.

Dyspnea shares many clinical and neurophysiological features with pain [1,2] but, as opposed to pain, dyspnea reflects a critical threat to a vital function, breathing. It's terrifying experiential dimension often surpasses that of pain, instilling a profound fear of dying, as patients relate: "when the shortness of breath was at its extreme, I thought I was going to die…I did have thoughts about suicide…" [3]. The association of fear of dying with the inability to verbally communicate (i.e. helplessness) and to control the ventilator (i.e. powerlessness), makes dyspnea a strong predictor of posttraumatic stress disorders (PTSD) in patients undergoing IMV (Fig. 1). Dyspnea relief should therefore be a major goal of care for ICU stakeholders, whose mission is to relieve symptoms and

[a]Sorbonne Université, INSERM, UMRS1158 Neurophysiologie Respiratoire Expérimentale et Clinique and [b]AP-HP, Groupe Hospitalier Universitaire APHP-Sorbonne Université, site Pitié-Salpêtrière, Service de Médecine Intensive – Réanimation, Département R3S, Paris, France

Correspondence to Maxens Decavèle, MD, PhD, AP-HP, Groupe Hospitalier Universitaire APHP-Sorbonne Université, site Pitié-Salpêtrière, Service de Médecine Intensive, Réanimation, Département R3S, F-75013 Paris, France. Tel: +33 1 84 82 79 39; e-mail: maxens.decavele@aphp.fr

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KEY POINTS

- Dyspnea in invasively mechanically ventilated (IMV) patients is frequent (50%), perceived as intense (50% of a full rating scale) and associated with inappropriate ventilator settings.
- Dyspnea is an independent predictor of posttraumatic stress disorder.
- Respiratory-related brain suffering (RRBS) refers to brain abnormal responses to respiratory-related messages and could be present in noncommunicative patients.
- Mechanical ventilation – respiratory distress observation scale (MV-RDOS) is the only available clinical surrogate of dyspnea or RRBS for IMV patients.
- Nonpharmacological treatments are strongly efficient to relieve dyspnea in IMV patients.

improve patients' comfort as well as treating the underlying disease.

The aim of this review is to describe and summarize current data regarding the prevalence, risk factors, consequences, assessment and treatment of dyspnea in critically ill patients undergoing IMV.

DEFINITION, PREVALENCE AND INTENSITY OF DYSPNEA

Definition: the 2024 ERS/ESICM statement

In a recent statement endorsed by the European Respiratory Society (ERS) and European Society of Intensive Care Medicine (ESICM), dyspnea is officially defined by "the symptom that conveys an upsetting or distressing experience of breathing awareness" [4▪▪]. The statement emphasizes that, as with other symptoms such as pain, dyspnea *per se* can only be perceived by the person experiencing it. However, most of IMV patients are unable to communicate (so called noncommunicative patients) and hence self-report the various forms of suffering that they experience, like pain, anxiety, thirst and dyspnea. Unfortunately, the inability to communicate does not negate the possibility of experiencing suffering. For this reason, experts panel defined the concept of respiratory-related brain suffering (RRBS) that refers to an ensemble of brain responses to abnormal or abnormally interpreted respiratory-related messages. The RRBS can be identified through self-reporting dyspnea by communicative patients, but a clinical appearance of unconsciousness/noncommunicative state cannot exclude RRBS as it is the case with

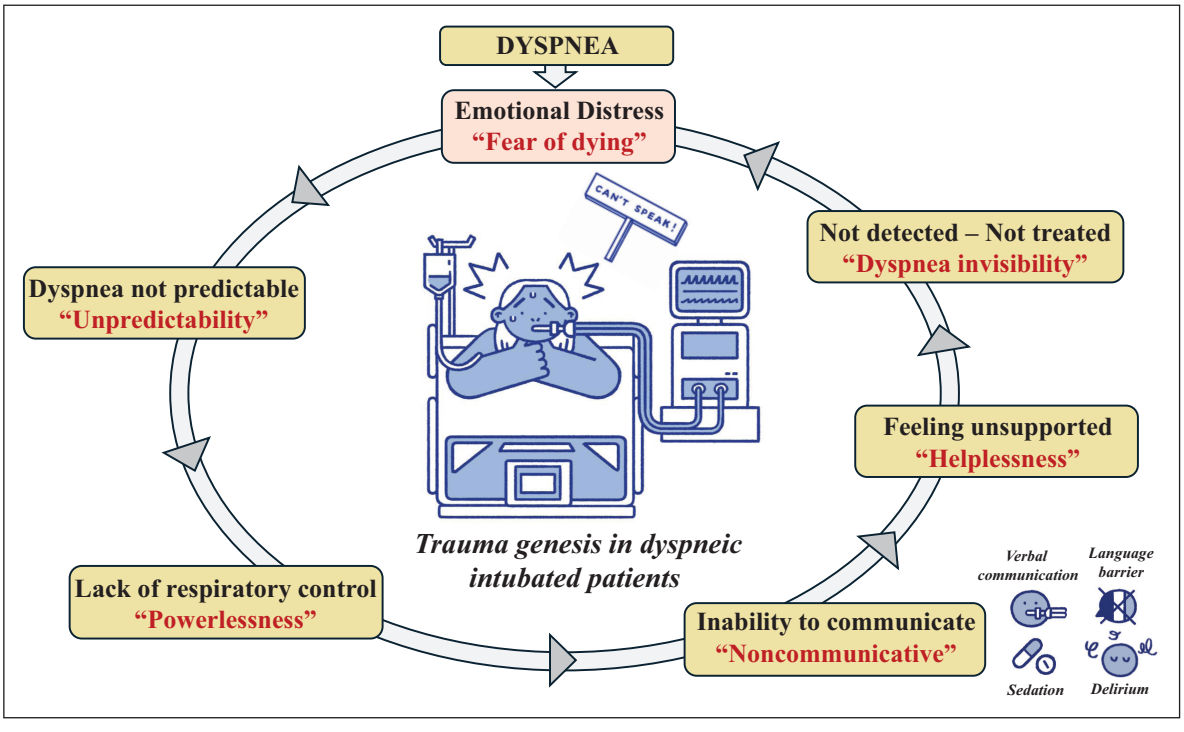

FIGURE 1. Psychogenesis of trauma in dyspneic patients receiving invasive mechanical ventilation (IMV). The association between dyspnea and trauma is explained by the fact that, dyspnea during IMV shares at least three key features with trauma: (1) intense fear of death, (2) unpredictability, and (3) powerlessness/lack of control, as patients are unable to escape or modify the source of dyspnea (the ventilator) and are often unable to communicate their suffering to stakeholders.

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nociception/pain [5]. This RRBS can be detected in noncommunicative patients using a standardized assessment of clinical signs (dyspnea observation scales).

There three main distinct dyspnea sensations are air hunger, generally associated with increased respiratory drive (hypercapnia), which is the most unpleasant of these sensations, labored breathing observed in case of increased respiratory muscle load, and chest tightness, more likely to be associated with bronchoconstriction. These three sensations can be observed in IMV patients, often combined [6,7].

Prevalence and intensity

The most robust epidemiological data regarding dyspnea epidemiology in IMV patients has been provided by a recent French observational prospective multicenter analysis of 612 IMV patients. In this study, dyspnea was measured as soon as patients were able to communicate (first awakening), and was reported by 35% of patients, rated 50 (40–70) % of a full dyspnea rating scale from zero (no dyspnea) to 100% (worst imaginable dyspnea) [7]. Overall, dyspnea is listed as a main outcome of approximatively 50 studies including about 20 prospective observational studies summarized in Table 1.

Table 1. Prospective observational studies in mechanically ventilated patients

Studies	*N*	Timing of dyspnea assessment	Dyspnea prevalence (%)	Dyspnea intensity (%FS)
Lush *et al.* [54]	5	Repeated every 4 h	100	Around 30
Moody *et al.* [23]	27	Repeated every 7 days	-	Extubation success: 35 ± 17 Extubation failure: 49 ± 20
Powers *et al.* [55]	28	Twice, 30-min interval	50	52 ± 17
Nelson *et al.* [56]	50	Repeated, daily recall of the last 24 h	Moderate: 17 Severe: 17	–
Karampela *et al.* [57]	238	Repeated every 4 h for 3 days	Mild: 8% Moderate: 2% Severe: 1%	–
Twibell *et al.* [58]	68	Once, before SBT	–	Overall: 35 ± 28 Extubation success: 35 ± 30 Extubation failure: 37 ± 22
Nelson *et al.* [59]	50	Repeated during SBT	–	Before SBT: 20 (20–55) End of SBT: 60 (40–80)
Wysocki *et al.* [60]	46	First min of SBT	-	45
Puntillo *et al.* [13]	171	Daily recall of the last 24 h	43	Intensity: 63 ± 3 (% FS) Distress: 78 ± 3 (% FS)
Vitacca *et al.* [61]	8	Repeated during SBT	–	Before SBT: 37 ± 32 End of SBT: 60 ± 35
Persichini *et al.* [14]	220	Once	68	42 (29–60)
Haugdahl *et al.* [19]	100	End of SBT	62	50 (20–70)
Chen *et al.* [20]	67	End of SBT	–	Weaning success: 10 ± 18 Weaning failure: 45 ± 38
Binks *et al.* [33]	30	Once	–	40 (40–70)
Gentzler *et al.* [32]	138	Once	47	49 (30–70)
Mazeraud *et al.* [62]	391	Once	-	50 (0–60)
Demoule *et al.* [7]	612	First awakening	34	50 (40–70)
Bureau *et al.* [21]	31	Beginning and end of SBT	–	Beginning SBT: 20 (0–40) End of SBT: 40 (0–100)
Saltnes-Lillegard *et al.* [63]	353	Daily assessment (7 days)	Admission 32 During ICU stay 36	Admission 56 (mean) During ICU stay 53 (mean)
Decavèle *et al.* [36▪▪]	50	Repeated assessments after dyspnea treatments	–	Baseline: 60 (39–70) After adjustments: 34 (20–52)

FS, full scale; ICU, intensive care unit; *N*, number of patients; SBT, spontaneous breathing trial.

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Pooling all these studies, about 50% of IMV patients report dyspnea, with an intensity rated about 50% of a full dyspnea rating scale [4▪▪,8]. As a comparison, similar pain ratings would probably be judged unacceptable by stakeholders and constitute guidelines threshold for prompt analgesia [9].

CONSEQUENCES OF DYSPNEA IN INVASIVELY MECHANICALLY VENTILATED PATIENTS

Immediate fear and posttraumatic stress disorders

One-half of ICU survivors who had been intubated during their ICU stay reported having experienced anxiety, fear, panic or insecurity during mechanical ventilation [10–12]. Dyspnea is generally perceived as the most distressing symptom in ICU patients [13]. Consequently, a significant association is demonstrated in various studies between dyspnea and anxiety in IMV patients [6,7,14,15]. In a report of 612 IMV patients, the prevalence of anxiety was three-fold higher in dyspneic patients than among nondyspneic patients (72% vs. 26%) [7]. In addition, dyspnea contributes to the pathogenesis of PTSD, a well known complication that affects about 15% of ICU survivors. In a prospective multicenter cohort on 612 IMV patients interviewed three months after ICU discharge, the proportion of patients with probable PTSD was higher in patients who experienced dyspnea while they were undergoing IMV than in those who did not (29% vs. 13%). Of notice, the cumulative exposition to dyspnea was independently associated with the occurrence of PTSD [7]. This association between dyspnea and the consequence of trauma is not unexpected, as dyspnea during IMV shares at least three key features with trauma [16]: intense fear of death, unpredictability, "Sometimes I sensed breathlessness coming, but most of the time it was unpredictable" [17], and powerlessness/lack of control, as patients are unable to escape or modify the source of dyspnea (the ventilator) and are often unable to communicate their suffering to caregivers: "I felt trapped" [18].

Dyspnea is a warning sign

Because dyspnea may result from respiratory system load-capacity imbalance, which is one of the major determinants of weaning outcome, dyspnea is associated with weaning failure [6,15,19–23]. Dyspnea two hours after extubation is also a strong predictor of reintubation [24]. For the same reason, in patients admitted for acute respiratory failure and managed with standard oxygen, noninvasive ventilation or high flow oxygen therapy, moderate to severe dyspnea is associated with a higher risk of intubation [25▪].

Although dyspnea is strongly associated with mortality in various settings such as at hospital admission [26], after myocardial ischemia [27] or in chronic obstructive pulmonary disease patients [28], it has not been yet found a clear and significant association between dyspnea and mortality in patients undergoing IMV in the ICU [6,7,19]. However, in patients receiving noninvasive ventilation for an acute respiratory failure, the persistence of dyspnea after the first noninvasive ventilation session is independently associated with mortality [29].

For all these reasons, addressing dyspnea in IMV patients should be a crucial element of daily practice monitoring and care, but to be treated, dyspnea must be detected and measured.

DETECTION OF DYSPNEA AND RESPIRATORY-RELATED BRAIN SUFFERING

Experience shows that dyspnea is not routinely assessed in ICU patients, and very few centers systematically assess dyspnea at the time of hospital admission [30,31]. Unfortunately, the performance of caregivers to detect dyspnea in the patients they are in charge of is very poor [19,32,33]. For instance, when patients scored dyspnea 50 (20–70) % of a full dyspnea rating scale, physicians gave a score of 20 (10–40) % [19]. In addition, the detection of dyspnea is not systematically followed by a therapeutic response. In a prospective observational study, the detection of moderate-to-severe dyspnea by nurses was not associated with any therapeutic intervention. By contrast, when pain was detected, it was often followed by the administration of opioids [32]. The term "invisibility of dyspnea" describes these discordant evaluations of dyspnea between patients and stakeholders and the lack of response or even the avoidance of stakeholder, possibly due to the fact that they do not know how to relief dyspnea in their patients [34]

In communicative patients, the assessment of dyspnea simply consists in asking patients for the presence or absence of dyspnea by simple dichotomous trigger questions, such as "is your breathing comfortable? ", "do you feel breathless? ", "are you getting enough air? ", then asking them to self-report their dyspnea intensity (if dyspnea is detected) on a numerical rating scale (visual analog, numerical, Borg, categorical or Likert scales) [1].

Dyspnea observation scales may help detecting dyspnea or RRBS in noncommunicative patients. These scales infer the presence of dyspnea based

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on three dimensions of dyspnea: breathing (respiratory rate, use of neck muscles, abdominal paradox during inspiration), neurovegetative (heart rate) and emotional (facial expression of fear). The mechanical ventilation – respiratory distress observation scale (MV-RDOS), diagnoses RRBS and infers dyspnea in noncommunicative IMV patients with a MV-RDOS ≥3 predicting a dyspnea numerical rating scale (NRS) ≥3/10 with good performance [35–37].

MECHANISMS OF DYSPNEA OR WHY DYSPNEA OCCURS

Pathophysiology of dyspnea

Extensive reviews on the pathophysiology of dyspnea have already been published [2,4▪▪]. Briefly, it has been proposed that efferent neural drive to breathe containing the expected sensory consequences (the corollary discharge) is continuously compared within the cortex and the insula to the flux of respiratory-related afferents incoming from various receptors (e.g. chemoreceptors, lung stretch receptors and C-fibers, trigeminal nerve sensory branches). Dyspnea occurs in case of a mismatch between the expected (corollary discharge) and real (afferences) sensory information provided by the achieved ventilation.

As illustrated in Fig. 2, dyspnea results from an imbalance between the "demand for breathing" (respiratory drive) and the "capacity of the respiratory system to satisfy this demand". The "capacity" of the respiratory system depends on the respiratory muscles function and the impedance of the respiratory system. In IMV patients, this "capacity" depends also on the level of assistance that the ventilator provides (Fig. 2).

Determinants of the respiratory drive (Fig. 2, left column). IMV patients are exposed to many sources of increased respiratory drive such as acidosis either related to increased blood CO_2 level (increased CO_2 production or retention) or decreased base excess (metabolic acidosis). Hypoxemia, anemia, fever or, suprapontine factors such as anxiety (helplessness, fear of dying), mood state [7], or tolerance to suffering, can also influence the respiratory drive to breathe.

Determinants of the capacities of the respiratory system (Fig. 2, middle column). In IMV patients, the

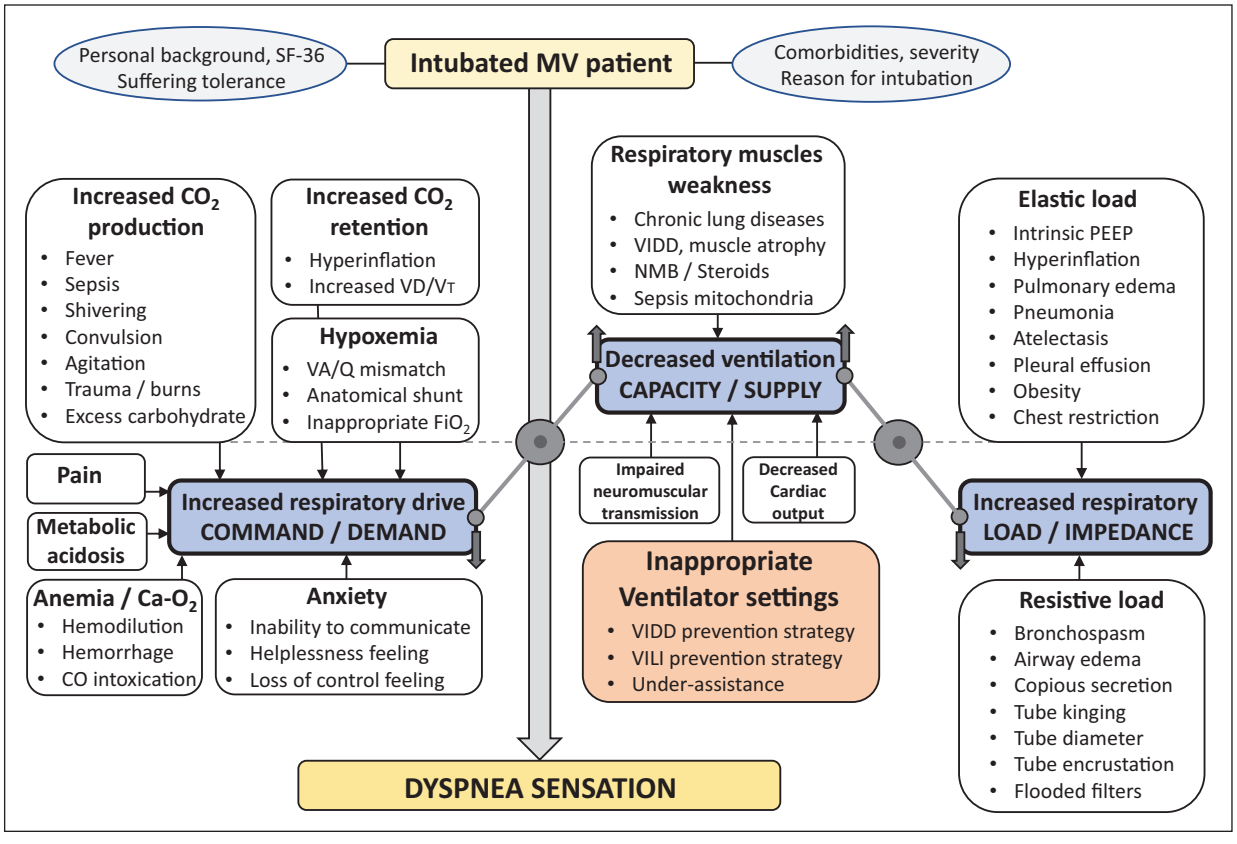

FIGURE 2. Pathophysiology of dyspnea in invasively mechanically ventilated patients. Ca-O_2, oxygen content of arterial blood; CO, carbon monoxide; FiO_2, fraction of inspired oxygen; MV, mechanically ventilated; PEEP, positive end-expiratory pressure; VA/Q, alveolar ventilation / perfusion ration; VD/VT, dead volume/tidal volume; VIDD, ventilator-induced diaphragm dysfunction; VILI, ventilator-induced lung injury.

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capacities of the respiratory system are likely to be altered because of the presence of underlying chronic lung diseases, ventilator induced diaphragm dysfunction (disuse atrophy), ICU-acquired weakness but also because of cardiocirculatory dysfunction.

In IMV patients whereas mechanical ventilation is supposed to unload respiratory muscle and hence to reduce load capacity imbalance, it is worth mentioning that inappropriate ventilator settings are major determinants of dyspnea (Fig. 2). Assist control ventilation mode is associated with increased risk of dyspnea [6], especially when inspiratory flow is set below 40 L/min [6,38] with low tidal volumes [39]. With pressure support ventilation, "reasonable" increase in pressure support is generally associated with a decrease in dyspnea [40,41]. However, over-assist may also cause dyspnea and data support a U-shaped relationship between the level of assistance, the subsequent tidal volume generated and dyspnea; when assistance is either too high or too low, dyspnea increases [36▪▪,42,43] (Fig. 3). Similar U-shaped relation as also been observed with the

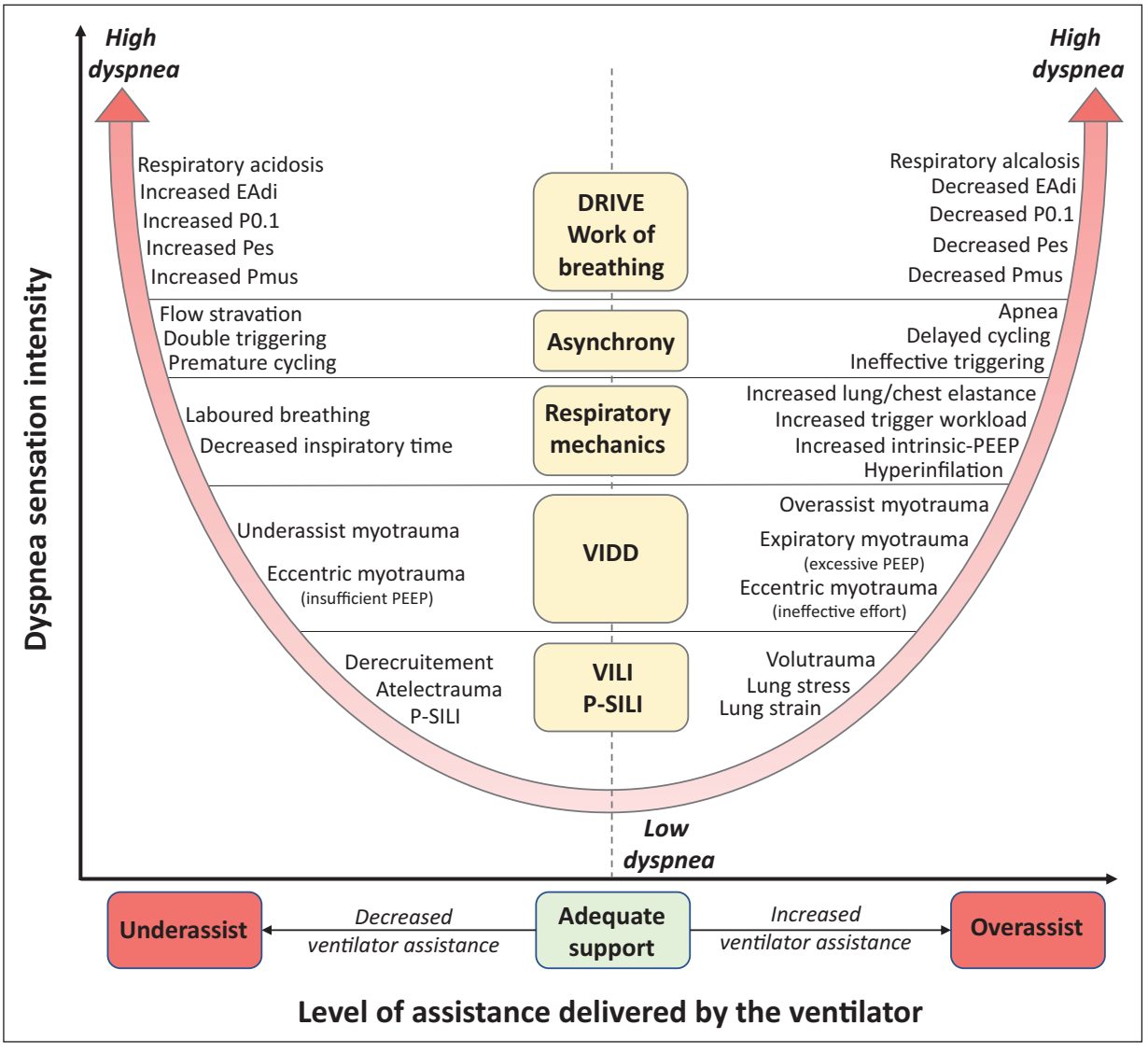

FIGURE 3. Relation between dyspnea sensation and level of ventilatory assistance. Red U-shaped arrow traduces that when ventilatory assistance level is either too high or too low, dyspnea or respiratory-related brain suffering may increase. Inside yellow boxes traduce the modifications in drive, patient-ventilator asynchronies, respiratory mechanics, VIDD and VILI that could be observed following ventilatory assistance level modification, that should be also considered the clinical decision making of ventilator settings adjustments. EAdi, diaphragm electrical activity; P0.1, airway occlusion pressure; PEEP, positive end-expiratory pressure; Pes, inspiratory swing in esophageal pressure; Pmusc, the pressure generated by the respiratory muscles to inflate both the lung and the chest wall; P-SILI, patient self-inflicted lung injury; VIDD, ventilator-induced diaphragm dysfunction; VILI, ventilator-induced lung injury.

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inspiratory flow slope in pressure support mode [44]. Decreasing expiratory triggering (delayed cycling) in patients with increased respiratory system elastance reduces inspiratory muscle workload, patient-ventilator asynchronies (double triggering) and dyspnea [45]. Positive-end expiratory pressure (PEEP) adjustments may also reduce dyspnea by offsetting respiratory load generated by intrinsic-PEEP [46] and increasing end-expiratory lung volume [47] which stimulates lung stretch receptors (Fig. 3).

*Determinants of the load of the respiratory system (Fig. 2, right column).*Beside altered respiratory mechanics like elevated resistance (lung obstructive diseases) and low compliance (acute respiratory distress syndrome), which are major determinants of dyspnea, healthcare-related activities (e.g. bathing, positioning), also act as loads to the respiratory system and contribute to dyspnea occurrences especially during respiratory procedures such as ventilator disconnection, tracheal suctioning, or fiberoptic bronchoscopy [13].

Understanding the pathophysiological mechanism of dyspnea is essential to build a therapeutic strategy aiming at relieving dyspnea in patient.

TREATMENT OF DYSPNEA OR RESPIRATORY-RELATED BRAIN SUFFERING

The relief of dyspnea raises moral and ethical question. Since asphyxiation (i.e. dyspnea) can induce more severe suffering than other forms of physical torture, it is reasonable to argue that the proper assessment and treatment of dyspnea in IMV patients is a human right [16]. By analogy with pain, or based on more physiological considerations [48▪], a dyspnea-NRS >3/10 is considered as clinically important threshold and should prompt stakeholder to look for dyspnea risk factors and to relieve dyspnea.

Once dyspnea is diagnosed, it is essential to adopt a step-by-step procedure to identify the cause of dyspnea and treat that cause whenever possible (Fig. 4). By analogy with pain, achieving a dyspnea-NRS ≤ 3 seems a reasonable objective to tailor treatment [4▪▪].

Communication: First step is to offer to the patient active listening and understanding, a directed empathetic and reassuring approach, which fight against an existential feeling of helplessness [49].

Circuit/technical emergency: The second step is look for a rapidly reversible cause of dyspnea such as an obstructed ventilator circuit, flooded filters, copious tracheobronchial secretions, and to improve position in the bed.

Ventilator settings adjustment: The third step is to optimize ventilator settings, like increasing a too low inspiratory flow [38] in assist control ventilation mode, switching to pressure support ventilation mode [6], adjusting the inspiratory pressure support level and the expiratory trigger [36▪▪,42,45]. Nourishing sensory cortex with respiratory-related afferences (e.g. high tidal volume, high inspiratory flow rate) strongly mitigates dyspnea sensation. Ventilators settings adjustment may also have positive impact on gas exchange (e.g. decrease in $PaCO_2$), which in turn reduce respiratory drive and dyspnea.

Fooling sensory cortex with extra-thoracic afferences: If clinically important dyspnea persists, various nonpharmacological measures to stimulate extra-thoracic sensory afferents such as opening a window, a mister, a fan or cold air applied to the face. The relevance of nonpharmacological intervention has recently been highlighted in a study including 48 dyspneic mechanically ventilated patients in whom dyspnea was efficiently reduced by applying a fan to the face [50▪▪].

Minimize the impedance of the respiratory system: In any cases, the identification of a nonresolutive pathophysiological process is mandatory and led to specific treatment such bronchodilators, pleural drainage, diuretics, corticosteroids or atelectasis removal [6].

Nonrespiratory causes of increased respiratory drive: The sixth step is to consider the treatment of a profound metabolic acidosis, an anemia, or fever.

Silencing/modulating insular cortex: If clinically important dyspnea persists, it is possible to efficiently modulate the emotional component of dyspnea by using relaxing music [50▪▪] or hypnosis [51▪]. Music exerts its positive emotional effects through cognitive distraction but probably also through the secretion of endorphins [52].

Pharmacological interventions: Finally, if clinically important dyspnea persists, morphine titration (maximum 10 mg morphine chlorhydrate) has been demonstrated efficient to relieve dyspnea in dyspneic IMV patients [36▪▪]. Benzodiazepines, relieving anxiety, reduce dyspnea [53] even not specifically tested in IMV patients. Nebulized diuretics to reduce dyspnea has never been tested in IMV patients.

CONCLUSION

In intubated patients, dyspnea is common, often severe, and linked to adverse outcomes like distress, prolonged weaning, and posttraumatic stress disorders. It frequently stems from inappropriate ventilator settings. Even in noncommunicative patients, RRBS can be detected using observation scales. While pharmacological treatments exist, nonpharmacological interventions, such as adjusting ventilator

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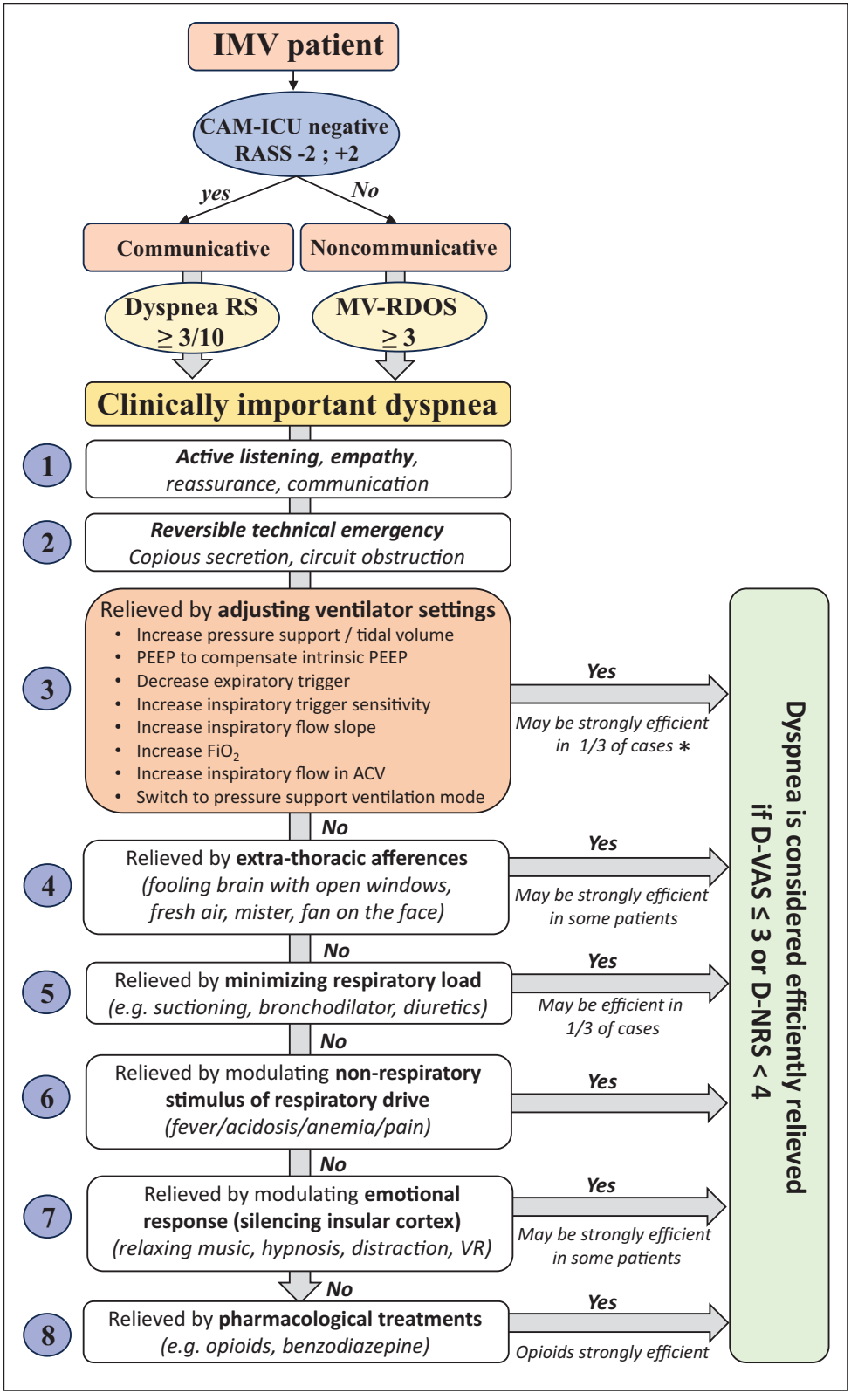

FIGURE 4. Algorithm for the diagnosis and treatment of dyspnea in intubated patients. IMV, invasively mechanically ventilated; MV-RDOS, mechanical ventilation – respiratory distress observation scale; RS, rating scales, D-VAS, dyspnea visual analog scale; D-NRS, dyspnea numerical rating scale; PEEP, positive end-expiratory pressure; ACV, assist control ventilation mode; FiO_2, fraction of inspired oxygen; RASS, Richmond Agitation and Sedation Scale, CAM-ICU, Confusion Assessment Method for the ICU. *Ventilator settings adjustments proposed here do not correspond to all patient-centered situations and must adapted to each patient.

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settings, can effectively reduce dyspnea. Despite its impact, dyspnea is often underestimated in clinical practice. Systematic assessment, akin to pain evaluation, could help manage RRBS and reduce psychological consequences. Techniques like music, hypnosis, and sensory stimulation show promise in treating its affective and sensory aspects.

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Conflicts of interest

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Maxens Decavèle reports personal fees from ISIS medical (congress registration fees).
Gabriel Kemoun reports no conflicts of interest.

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